Because our field wants periodic limb movements of sleep (PLMs) to be a “sleep disorder,” the controversy over the clinical importance (or lack thereof) of PLMs continues to smolder on – hanging around like a bad smell. It is intuitive: blame PLMs for insomnia in patients with both, blame PLMs for excessive daytime sleepiness (EDS) in patients with both, or dismiss PLMs as a variant of normal in asymptomatic patients. The clinical problem has been given a label, and we can go on to the next case. Convenient – we have just had it all 3 ways. This approach is handy and comforting as long as we don’t let ourselves be bothered by the pesky fact that despite extensive and exhaustive attempts, our field has failed to provide one shred of scientific evidence that PLMs have any predictable clinical consequence.

Periodic limb movements of sleep (PLMs) are rhythmical extensions of the great toe and dorsiflexion of the ankle, knee, and hip (and probably represent spontaneously occurring Babinski signs or triple spinal flexion reflexes - normal phenomena during the lighter stages of NREM sleep due to enhanced spinal cord excitability) lasting 2-4 seconds with a frequency of one every 20-40 seconds. PLMs were first recorded by Lugaresi. Scoring criteria were defined by Coleman in 1982, and later modified by the American Sleep Disorders Association and codified in the International Classification of Sleep Disorders. Simply by defining the polysomnographic (PSG) features of PLMs, a sleep “disorder” was created.

There is a great deal of confusion between PLMs and the restless legs syndrome (RLS). Up to 90% of patients with the clinical symptom of RLS will display the PSG observation of PLMs. The converse is not true – the overwhelming majority of patients with PLMs on PSG do not have RLS symptoms. Even in patients with RLS who display PLMs, the severity of RLS symptoms do not correlate with PLMs frequency.

Arbitrarily, and without documented clinical correlation, a PLMs index >5/hr is said to be “abnormal”. Using the arbitrarily determined definition of “abnormality” of a PLMs index >5/hr, 30%-86% of adults aged 60 years or older are “abnormal.” Then there are the sticky issues of the striking night-to-night variability, and the arbitrary amplitude and sequence criteria. How many nights of monitoring are necessary, and what are the amplitude and frequency criteria?

It has been said that PLMs with “arousal” are clinically significant. The common misperception that the leg movements cause arousal is erroneous; a recent study indicated that 49% of the EEG arousals occurred before the leg movement, 31% simultaneous with, and in only 23% did the leg movement precede the EEG arousal.

Our field has yet to determine what constitutes an arousal, or what arousal index may result in clinical symptoms. The American Academy of Sleep Medicine arousal criteria have never been validated as to clinical significance. It may be that arousals measured by respiratory paradox or autonomic measures (increases in blood pressure or heart rate) without EEG evidence of arousal (by any definition) may be significant. It is likely that either/or both the EEG arousals and or PLMs are the manifestations of a common periodic central nervous system generator. PLMs are likely a manifestation of the cyclic alternating pattern, a microstructural sleep pattern of normal arousal instability during NREM sleep. It very well may be that the periodic arousals associated with PLMs are primary, with the PLMs being a secondary and variable accompaniment. Figure 1 demonstrates severe PLMs with EEG evidence of arousal in an asymptomatic patient.

Editorial reviews of this topic have argued that no consistent or predictable subjective or objective consequences of PLMs – in terms of either insomnia or hypersomnia – have ever been demonstrated. Subsequent studies have buttressed that viewpoint. Based upon the fact that PLMs are not more prevalent in insomnia or hypersomnia than in controls, Montplaisir has concluded: “...the validity of PLM disorder as a distinct nosological entity is highly questionable and in our experience, a diagnosis of PLM disorder has no specific utility”.

In one recent large study 61 patients with a mean PLM arousal index of 41.8/hr (many with PLM arousal index of >50/hr) had no more wake/sleep complaints than the control group of 60 patients with no PLMs. Interestingly, it was concluded that clinical data were not predictive of PLMs, and that formal polysomnographic evaluation was “necessary to establish the diagnosis of PLMD in patients with insomnia or hypersomnia” – despite the fact that those with very frequent PLMs had absolutely no clinical com-
In another study, arousals associated with PLMs were associated with lower sleep efficiency, higher percentage of stages 1 and 2 NREM sleep, and lower percentages of stages 3 and 4 NREM and REM sleep. Although these PSG observations were not associated with any identifiable subjective daytime symptoms, it was concluded that PLMs associated with arousal were “markers of disturbed sleep.”

In support of the concept that PLMs are of no clinical significance is the fact that there have been no valid studies documenting that treating isolated PLMs per se improves either nighttime sleep or daytime functioning in any condition. As a corollary, in patients with narcolepsy, the reduction of PLMs by bromocriptine does not improve the nocturnal sleep disruption in that disorder.

The PLMs controversy continues to smolder because our field wants PLMs to be significant, not because PLMs are significant. It is an intuitive and clinically convenient diagnosis unsupported by any scientific evidence. Most clinically significant medical phenomena eventually declare themselves during 20 years of intensive study. (It did not take 20 years and hundreds of studies to determine that penicillin was good for Gonorrhea.) Clinical significance of PLMs has been exhaustively sought – to no avail. It may be time to stop looking. Despite innumerable attempts, the clinical relevance of PLMs remains elusive. Once a diagnosis of PLMs has been made, clinical thinking ceases, and patients may be exposed to gratuitous medication. This is particularly true in the pediatric population. It is likely that the only consequence of PLMs is to the bedpartner(s).

**CONCLUSIONS**


---

**Figure 1**—This 5-minute epoch contains 18 PLMs. The overall PLM index was 91/hr (548 PLMs over 364 minutes of total sleep time) in this asymptomatic patient. Note numerous EEG arousals associated with PLMs. (LOC – left outer canthus, ROC – right outer canthus, A1/A2 – left/right mastoid, C3/C4 – left/right central EEG, O1/O2 – left/right occipital EEG, chin – submental EMG, Leg/L/Leg/R – left/right anterior tibialis EMG).